Adolescence is the time between the beginning of sexual maturation (also known as puberty, from the Latin pubertas, meaning adult) and the beginning of adulthood. Adolescence usually spans the years between ages 8 to 17 yr in girls and 11 to 19 in boys. Adolescence includes physical growth, sexual development, emotional, psychological, and mental change. Psychological maturation occurs as the child acquires adult-like behavior. During this period, adolescents are expected to become capable of adult behavior and response. Adolescence is a period of many transitions. During the teen years, adolescents experience changes in their physical development at a rate of speed unparalleled since infancy.

**Endocrine control:** The onset of puberty is signalled by the secretion of pulses of gonadotrophin-releasing hormone (GnRH). Prior to puberty various mechanisms suppress onset of puberty (both via hormonal feedback and central neural suppression of GnRH release). The occasional pulsatile GnRH secretion progresses to nocturnal secretion and on to 24-hour secretion. It has been postulated that the trigger for puberty comes from higher centers and is under partly neural control with genetics playing a part. For 2 years before puberty there is a rise in levels of adrenal androgens that can sometimes result in the early appearance of pubic hair and spots (Adnerache). This is also initiated by the nocturnal secretion of pulses of GnRH from the hypothalamus.

**Sexual development in girls:** Breast enlargement, occasionally initially unilateral, is the first obvious sign of puberty and occurs on average between 10 and 11 yr of age. Pubic and axillary hair growth in girls is a sign of adrenal androgen secretion. It starts at about the time of apocrine gland sweat production and the common complaint of axillary odour. Menarche usually occurs about 2-3 years after the start of breast development (thelarche). The median age of menarche is around 12 yr 6 mo in Indian girls. The growth spurt occurs early in female puberty. It is usually maximal (about 8 cm/year) during Tanner breast stages 2 and 3. But reduces to 4 cm/year at menarche. In the post menarcheal period maximum height gain remains around 5 cm only.

**Sexual development in boys:** Testicular enlargement usually occurs between ages of 12 and 13 years. The prepubertal testes are about 2 ml in volume, with puberty taken to begin when a volume of around 4 ml is attained. Penile and scrotal enlargement occur typically about a year after testicular enlargement. Pubic hair typically appear at a similar time. The growth spurt occurs later than in girls, possibly because testosterone is less of a stimulus to growth hormone responsiveness than estradiol in girls and is required in relatively higher concentrations of testosterone to produce the same anabolic effect. A greater and later growth spurt occurs in boys and ultimately achieves an average 12-13 cm greater height in adult men than the female counterparts. The growth spurt is on an average 2 yr later than girls and ceases only 1 year later.

Fig 1. shows stages of breast in girls and penis and testicular enlargement in boys (Sexual maturity rating 2-5 of Tanner).
Physical growth: includes rapid gains in height and weight. During a one-year growth spurt, boys and girls can gain an average 10-11 cm and 8-9 cm in height, respectively.

Adolescence Growth - Period extends for 2.5 to 3 years; to cross Sexual Maturity stages 2-5. Height gain is 27-29 cm in boys & 24-26 cm in girls. Weight gain in both being 25-30 kg. Weight gain results from increased muscle development in boys and body fat in girls.

Fig 2. Shows importance of correlating assessment of growth (height and weight), in adolescence with sexual maturity stages.
This understanding of sexual maturity to physical growth is important as in usual practice anthropologist and medical practitioners use the distant growth curves in relation to age (Fig 3, see example of 14 yr boy showing median height in as 158 and in SMR 2-5 height values are 151, 156.5, 162 and 166 cm respectively)
Brain growth and development in adolescence

Studies on the brain during the last decade show that it—along with height, weight and hormones—goes through dramatic changes during the middle school years. While outward changes are easy to see, brain development goes much deeper, but it can go far in explaining how and why your child does what he does.

According to the National Institute on Mental Health (NIMH), there is a surge of production of the brain's gray matter prior to puberty. Before this finding in 1999, it was thought that the brain overproduced gray matter only until about 18 months of age—after which there was a steady decline as unused brain cells were discarded. We now know that the area of brain growth during adolescence centers on the frontal lobe. This is the control center for "executive functions as planning, impulse control and reasoning".

The "Brain growth" is complex as "Gray matter" is made up of the cell bodies of neurons, the nerve fibers that project from them, and support cells. At birth each neuron has 2,500 synapse by 2 years, there are 15,000 synapses per neuron. At 3 years the first period of consolidation begins. This period tends to be characterized by children asking the question "why?" It is estimated that a four year old asks a "why" question every two and a half minutes. Around the age of six, there is a second surge as the brain starts to use language in increasingly complex ways. Up to the age of nine a child's brain continues to be twice as active as an adult's brain.

In adolescence brain adopts a "use-it-or-lose-it" pruning system, sloughing unused connections and increasing the speed of others. 30,000 synapses may be lost per second in the early adolescent brain leading to an ultimate loss of almost one half of the synapses. Areas of the brain responsible for executive functioning (such as strategic thinking, weighing risks and benefits and impulse control) continue to develop and refine connections through adolescence and into the mid-twenties.

Adolescents who engage in more dangerous activities have white-matter pathways that appear more mature than those of risk-averse youths. White matter is essentially the "wiring" the neural strands that connect the various gray-matter regions, where the actual nerve cells reside, that are otherwise independent of one another.

Maturation of white matter: a) increases the brain's processing speed; b) nerve impulses travel faster in mature white matter. Children who had been in an orphanage at any time in their lives had much smaller gray matter volume in the cortex of the brain and had smaller white matter volumethan those who had never been in an orphanage. Even if children were placed in loving foster homes, the formerly institutionalized children's gray matter didn't catch up. In contrast, white matter, however, seemed to be more resilient, as orphaned children placed in high-quality foster care had the same white matter volume as those who were never in an orphanage.

Children born of undernourished-anemic mothers and continued to live in "Endemic undernutrition" showed the intrauterine growth retarded offspring's of undernourished-anemic mothers have hypotonia, hypoexcitability, limp posture, shortening of sleep cycle, intra hemispheric asymmetry and abnormal paroxysmal discharges; suggesting dysmaturation of brain. These undernourished children followed from birth to preschool years had impaired intelligence, behavioral, conceptual and sensory motor development. During school age until 17.5 years follow up studies showed mobilization of amino acids from body muscles (increased serum enzyme activities i.e. LDH, ALP, AST, ALT, CK, CK-MB and CK-mm. 31- phosphorus magnetic resonancespectroscopy showed that -ATP and Pi in muscles was significantly increased at the cost of Pcr (Phosphocreatinine). Soft neurological signs - persisted. There was deficit in higher mental abilities. Brain MRI-frontal lobes showed reduction in size, and loss of asymmetry. Reaction time studies showed affects on perceptual abilities, information processing and analytical capabilities. It is important to note that early life undernourished children continued to have prolonged reaction time, even if they had attained normal nutrition.
In animal studies: The fetal brains of rat mothers fed wheat or Bengal gram (diet having limiting amino acid) showed, dissociation of brain growth (brain being more affected than the body). Further there were alterations in protein, glutamic acid and GABA metabolism; these were reversible to some extent on rehabilitation. In contrast, in latent iron deficiency fetal as well as weanling rat there was, irreversible decrease in brain iron content; GABA shunt enzymes, dopamine, norepinephrine and tyrosine (catecholamine metabolism) and in tryptophan, 5-hydroxy tryptophan and 5-hydroxy indoleacetic acid (5-hydroxytryptamine metabolism). The affects on neurotransmitter receptors during early stages of iron deficiency clearly indicate the deficits in both excitatory and inhibitory pathways of the central nervous system. Anemia nutritional or in thalassemia showed similar alteration on MRI studies e.g. similar iron content; increase in creatinine and aspartate and reduction in choline concentration.

Alcohol and the Teen Brain

Adults drink more frequently than teens, but when teens drink they tend to drink larger quantities than adults. There is evidence to suggest that the adolescent brain responds to alcohol differently than the adult brain, perhaps helping to explain the elevated risk of binge drinking in youth. Drinking in youth, and intense drinking are both risk factors for later alcohol dependence. Findings on the developing brain should help clarify the role of the changing brain in youthful drinking, and the relationship between youth drinking and the risk of addiction later in life.

REFERENCES


7. Read more: http://www.time.com/time/health/article/0,8599,1919663,00.html#tczz255hoe8Gfe